



# Epilepsy

# Epilepsy

- Chronic brain disease characterized by (unprovoked) repetitive epileptic seizures .
- Single epileptic seizure can be provoked also in a healthy human without neurologic disease! (sleep deprivation, abstention syndrome – barbiturates, alcohol, hypoglycemia, extremely strong stimulus...)
- One of the most frequent chronic brain diseases

# Etiology of epilepsy

- **Primary:** unknown cause, onset usually in the childhood or adolescence, genetic disposition
- **Secondary (symptomatic):** no genetic disposition, known epileptogenic factors
  - states after stroke, posttraumatic scar, tumors, inflammation, developmental brain disorders...

E.g. in 5-9% of patients after stroke vascular epilepsy develops.

**In general:** Any affection of neurons can lead to epilepsy.

**But:** No neurons = No epilepsy

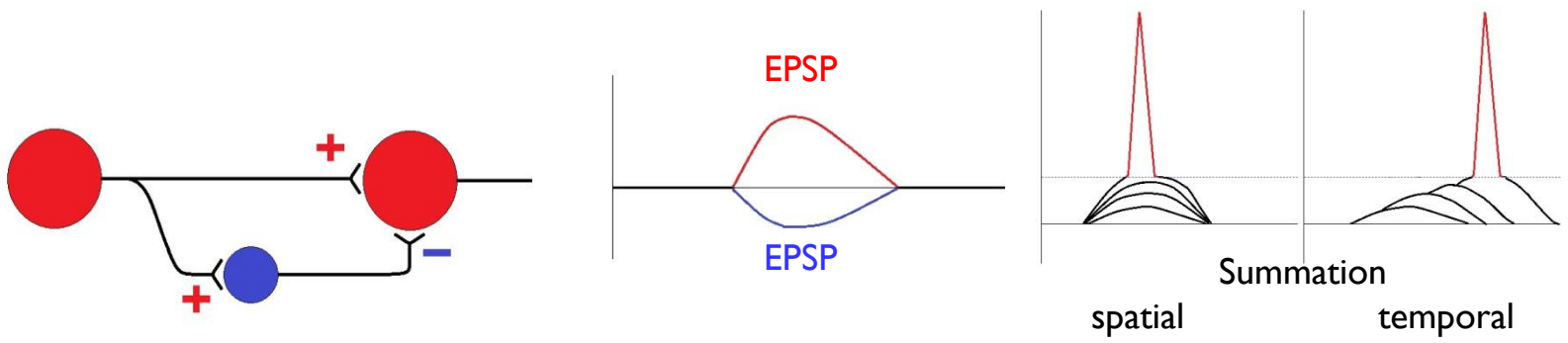
→ Epilepsy does not originate in the center of the scar or postmalatic pseudocyst, but from its margin or surrounding where injured neurons survive.

# Abnormal epileptiform behavior of neurons of the epileptic focus

- **Hyperexcitability** – abnormal excessive electric reaction of neurons to a common stimulus
- **Hypersynchrony** – ability of neuronal population of the focus to produce synchronic action potentials
- **Epileptic focus:** any area of the gray matter (neocortex, hippocampus...)

# What is the mechanism of epileptiform behavior of neurons?

- **Hyperexcitability of the brain cortex:** failure of labile physiological balance between excitatory glutamatergic and inhibitory GABAergic afferentation of the pyramidal cells
- **Existence of the thalamic pacemaker:** It forces its oscillative electric activity to the pyramidal cells of the cortex (NonREM sleep, absence)
- **Neuromodulatory effect of the brain stem RAS:** NA and serotonergic part of the RAS increases excitability of brain cortex neurons (sleep deprivation!)



Normal state

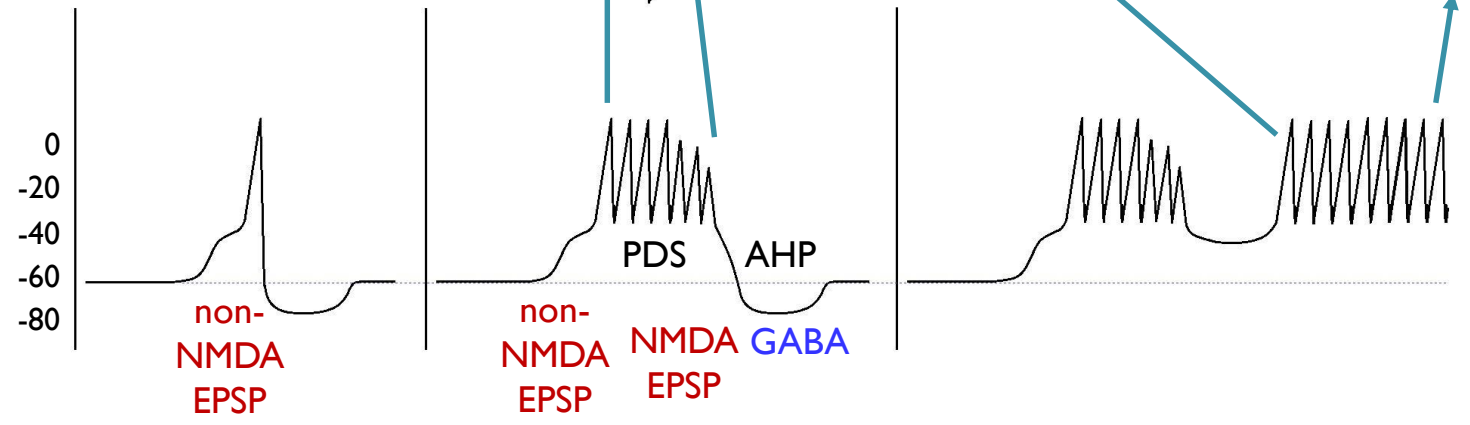
Epilepsy between seizures

Epileptic seizure

EEG



intracellular recording



- PDS = paroxysmal depolarizing shift
- AHP = afterhyperpolarization
- EPSP = excitatory postsynaptic potential
- IPSP = inhibitory postsynaptic potential

# Factors determining epileptic seizure rise

- **Epileptic focus**

- morphologically defined (tumor, scar, malformation...) or undefined

- **Threshold**

Determined by properties of the focus and recent state (sleep deprivation, glycemia, medicaments, drugs, alcohol...)

- **Stimulus**

- intensity and character (E.g. repetitive light flashes are dangerous)

# Classification of epileptic seizures (topographic point of view)

- **Partial seizures:** abnormal electric behavior of neurons in a restricted cortical area of one brain hemisphere

According to consciousness alteration: simplex partial seizures x complex partial seizures (qualitative disorders of consciousness)

- **Generalized seizures:** abnormal electric activity affects cortical neurons of both brain hemispheres in a diffuse way (primary x secondary, convulsive x nonconvulsive)



# Partial seizures

- epileptic focus in a restricted area of the brain cortex
- **simplex**: without changes of consciousness ( g. praecentralis → muscle contractions on contralateral side of the body, g. postcentralis → paresthesia on contralateral side of the body), Jackson's epilepsy
- **complex**: with deterioration of cognitive functions, often the frontal and temporal lobe, psychomotor seizure: running, senseless activity, changes of affects, instincts (amygdale, hippocampus)

# Generalized seizures

- **Primary** (the epileptic focus is the whole brain cortex from the beginning of the seizure) x **secondarily** generalized

**Absence** (petit mal), 5-15s many times per day

In children up to 10 years of age

The child looks stiff in front of itself and does not respond.

EEG: spike oscillations with a frequency of 3Hz

**Tonic-clonic seizure** (grand mal), 1-5min

Cramps, loss of consciousness, tongue biting, incontinency

After the seizure: confusion, aggressivity, amnesia

EEG: sharp waves with high amplitude, up to 100Hz

Complications: status epilepticus – glutamate excitotoxicity!

# Examination in interictal period

- Abnormal electric activity of the neurons can persist during the subclinic period.
- Examination after sleep deprivation, hyperventilation, photostimulation...
- Detection of the epileptic focus: EEG registration of epileptiform patterns (abnormalities):
  - spike – 20-70 ms, sharp wave – 70-200 ms
  - marked in the background EEG activity (focal x generalized)